

Endocrine (1)

1

- Thyroide (Graves, hashimotos)
- DM (How to Dx, Mx, Types, DKA, hypoglycemia, lactic acidosis)

* Introduction to Endocrine. *

Different b/w Endocrine

secreted hormones
direct into blood

ex:- Hypothalamus
Pituitary
Thyroid
Pancreas
Adrenal gland
♀ ovary
♂ testis

Exocrine

secret inside gland in duct
out side blood

ex:- Parotid salivary gland
sweat gland
Pancreas

→ hypothalamic pituitary axis

Hormones = glycoprotein substance.

Pancreas mixed gland

α , β , δ cells secrete hormone

cells secrete Pancreatic juice inside duct

* Hormones not under control hypothalamic pituitary axis :-

Aldosterone $\rightsquigarrow$ by renin angiotensinogen system stimulated by hypotension

Insulin $\rightsquigarrow$ stimulated by level of glucose

PTH $\rightsquigarrow$ by Ca^{2+} level

most common endocrine disease

1st common (Thyroid), 2nd common (DM)

3rd now a days (Adrenal insufficiency)

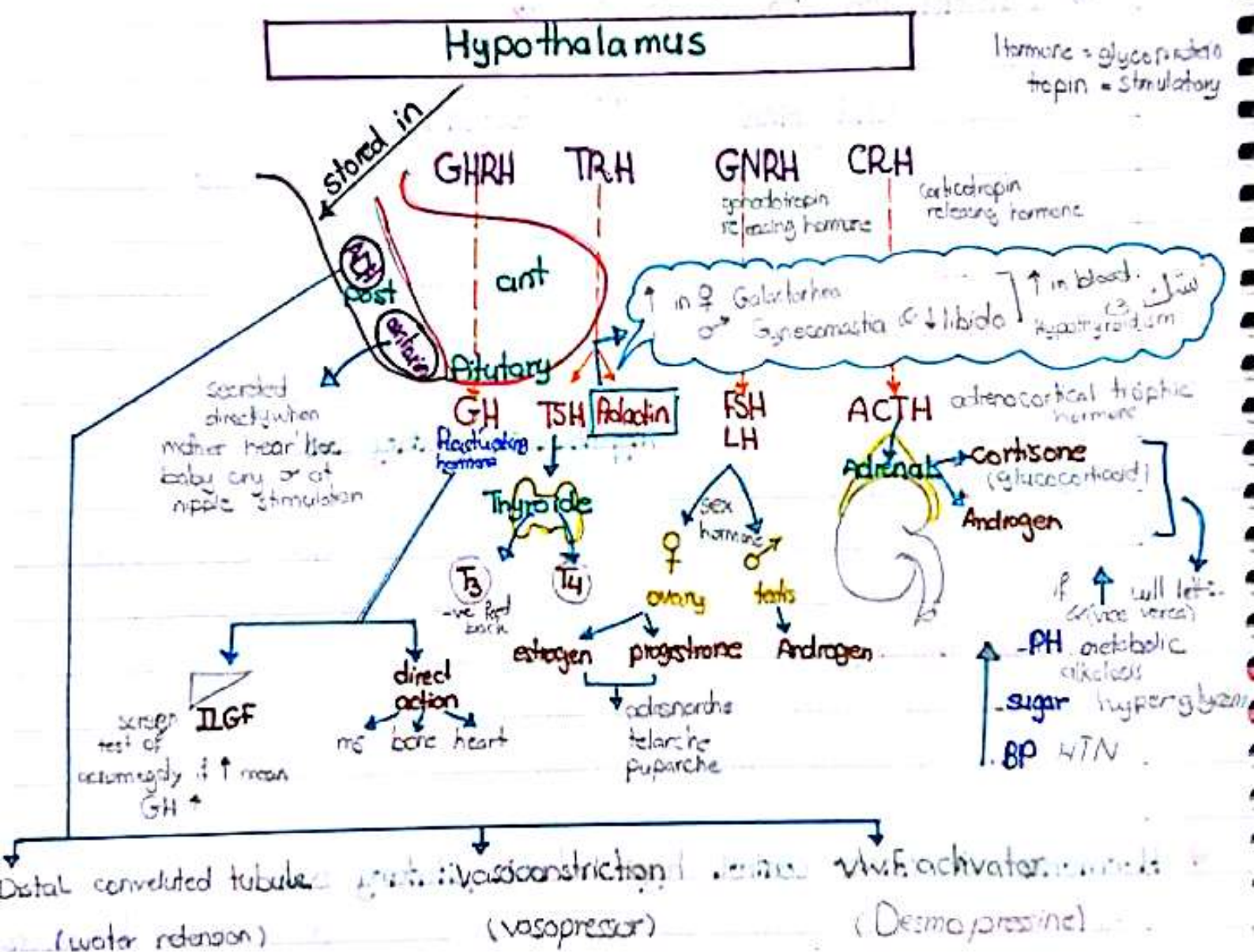
VISIT...

LANZAROTE
Caliente.COM

↑ incidence of adrenal insufficiency now a days d.t. ~~~~~

T.B infection

HIV infection



in TFT: Thyroid gland more accurate to measure **T₄** d.t. long half life in blood (7 days)

T₃ short half (hours)
active form

* Space volume & Loss :-

- 1st space (BVs) → BVs edema
 2nd space (cells) → intracellular edema
 3rd space (interstitial) → interstitial edema

* Endocrine disturbance *

1. Hormones ↑
2. Hormones ↓
3. Hormones resistance (Type II DM)
4. Hormones receptor hypersensitive to catecholamine → Palpitation
5. Endocrine gland mass Adenoma (Functional & non-functional)
 best dt → benign not converting into adenocarcinoma.

* Hormones ↑ :-

1^{ry} causes
(gland itself)

- Autoimmune (Graves)
- Adenoma Functional (single or multiple)
- hyperplasia
- Exogenous

2^{ry} causes
(Pituitary gland)

- Adenoma

3^{ry} cause
(hypothalamus)

* Hormones ↓ :-

- Autoimmune (Hashimoto's)
- resection gland & replacement
- excessive radiotherapy overdose

2^{ry}
Panhypopituitarism (tumour / trauma / Age)

at 1st (hyperthyroidism) then 2nd 3rd 4th 5th 6th 7th 8th 9th 10th 11th 12th 13th 14th 15th 16th 17th 18th 19th 20th 21st 22nd 23rd 24th 25th 26th 27th 28th 29th 30th 31st 32nd 33rd 34th 35th 36th 37th 38th 39th 40th 41st 42nd 43rd 44th 45th 46th 47th 48th 49th 50th 51st 52nd 53rd 54th 55th 56th 57th 58th 59th 60th 61st 62nd 63rd 64th 65th 66th 67th 68th 69th 70th 71st 72nd 73rd 74th 75th 76th 77th 78th 79th 80th 81st 82nd 83rd 84th 85th 86th 87th 88th 89th 90th 91st 92nd 93rd 94th 95th 96th 97th 98th 99th 100th

* organs have endocrine function :-

Heart	ANP, ADP
Kidney	Erythropoietin (80%)
Liver	Erythropoietin (20%)
adipose tissue	Androgen $\xrightarrow[\text{enzyme}]{\text{Aromatase}}$ Estrogen in fat

... Thyrotoxicosis have gynecomastia & $\rightarrow$ hyperactivity aromatase

* C/P of endocrine system :- "systemic manifestation"

D/D of endocrine disease (Lethargy & depression)

hypothyroidism / DM (I & II) / adrenal insufficiency / cushing

D/D of (wt gain)

hypothyroidism , cushing

D/D of (wt loss)

Thyrotoxicosis , DM

D/D of (Palpitation)

Thyrotoxicosis , Pheochromocytoma

D/D of , headach ,

Pituitary adenoma , Pheochromocytoma

* Investigation of endocrine :-

5

- Hormone Level : TFT, azurite

but in circadian rhythm hormones - cortisol $\uparrow$ at morning
(the measurement not accurate) - GH $\uparrow$ at night

Cushing ($\uparrow$ cortisol) all time

Acromegaly ($\uparrow$ GH) " "

- suppression test in pts who have $\uparrow$ in hormones

- stimulatory test

- U/S

- CT

- MRI

- Endoscopic US

- radioisotope scan in masses to differentiate b/w functional & non-functional
if +ve then do invasive & take biopsy

→ inject radioiodine if :-

$\uparrow$ uptake (hot) functional gland

Graves, single nodule, multiple nodule

$\downarrow$ uptake (cold) Non functional gland

→ take biopsy

* case :-

TSH $\downarrow$
T₃, T₄ $\uparrow$
Isotope scan (N)

$\uparrow$ in thyroid hormones

exogenous
thyroxine
intake

stroma ovary

secrete external

Thyroid hormone

الخلايا
الغدية

Endocrine 2

①

brood term

Hyperthyroidism (↑ hormone from gland or exogenous)

.. Thyroid ..

specific term

Hyperthyroidism (↑ hormone from self)

Hypothyroidism

↑ size of thyroid gland regarding function
Goiter

Graves

Autoimmune
♀ > Ab ↑ stimulatory

Hashimoto's

Autoimmune
♀ > Ab destructive

diffuse goiter

diffuse goiter

soft in consistency

↓
Hyper Hypo evo
* to Dx :-
(symptoms / sign / TFT.)

* D/D of diffuse goiter :-

- ① simple diffuse goiter (↑ demand) in (puberty & pregnancy) :-
- ② Graves disease
- ③ Hashimoto's

* causes of hyperthyroidism :-

- %75 Graves (Exophthalmus)
- %14 multiple toxic nodule
- %5 single toxic nodule

Mx by :- surgical therapy + steroid

Graves

Exophthalmus

أم آلتوم

Tremor

نضارات بين ادرق

اعطوها دواء معاشاش

حالة - مفروض بيروا علية

* Euthyroid graves disease :-

ومكان الحيلة جنب vocal cord

on ↓ treatment
compensate the
↑ of thyroid
hormone

Exophthalmus

↑ vocal cord palsy 1%

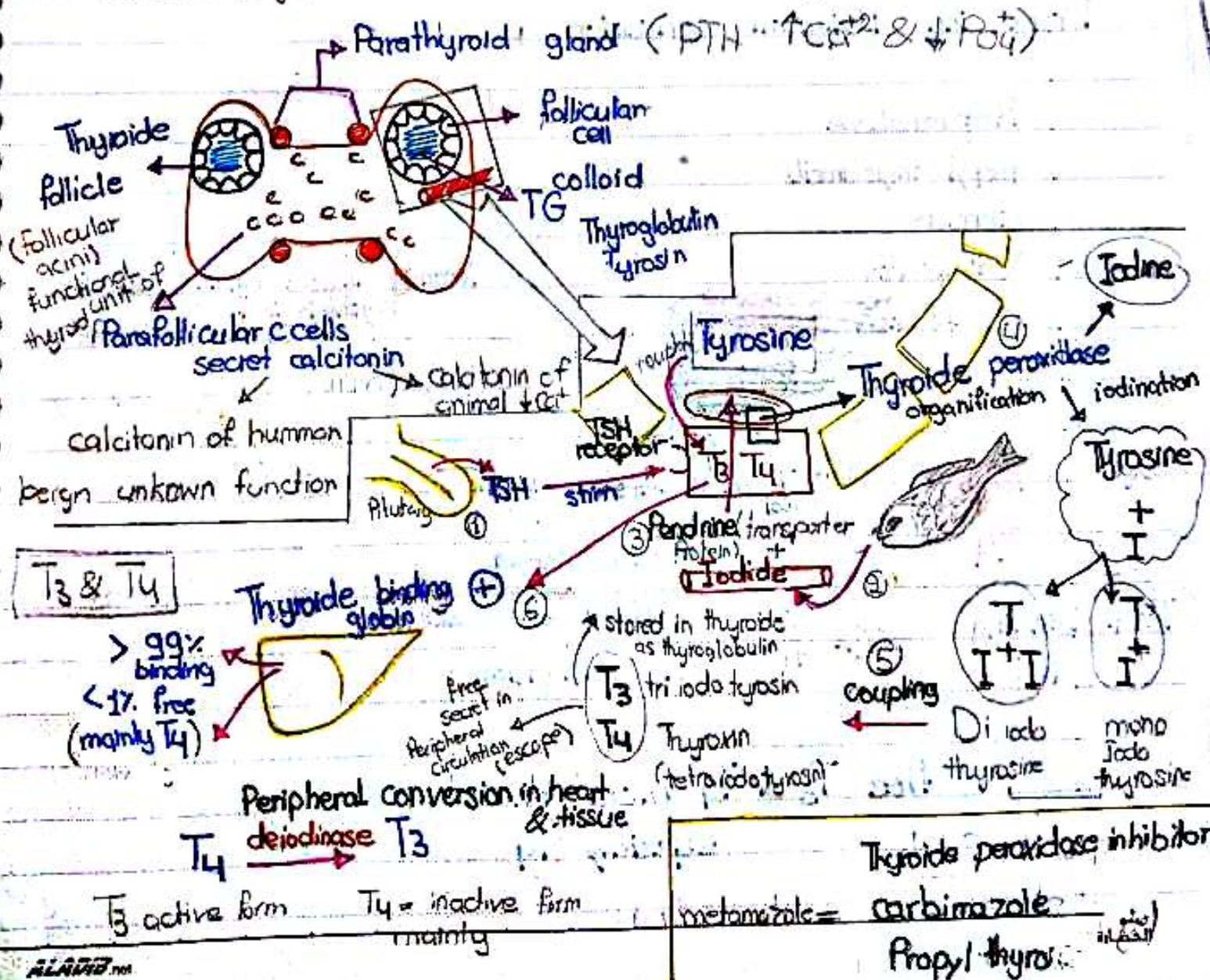
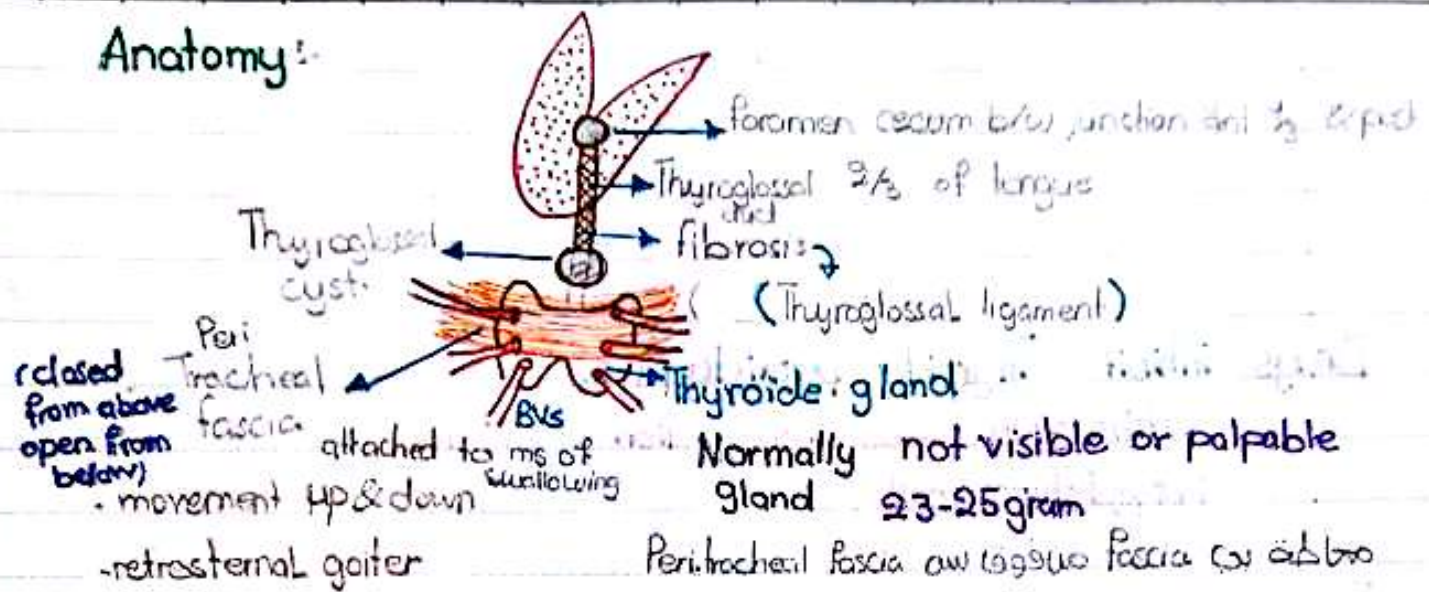
still need surgery to correct

Proptosis

radiotherapy
the condition

C/I in exophthalmus cases worsening

Anatomy:



Pendred S. - Pendred congenital absent
mental retardation / dwarfism / deafness / mutasim

Drugs inhibit Thyroide peroxidase..

Carbimazole (more strong action) 1st line except pregnancy & Breast feeding
Propyl thyouracil

Drugs inh Peripheral conversion:-

توقف الاعراض
immediate



- Propranolol
- propyl thyouracil
- Steroids
- Na^+ ipodate
- K^+ ipodate

Normally Thyroid binding globulin
synthesis in liver
not secreted in urine



liver cirrhosis



nephrotic



>99% binding
Thyroid Binding globulin
high molecular wt
in OCP or pregnant ♀
(falsly high)

<1% free

Total = binding + free

Total not accurate
free accurate (specific)
dt total affctd by other disease
(falsly Low)

free T_3 & T_4 more accurate
especially T_4

* TFT (Thyroid function test) :-

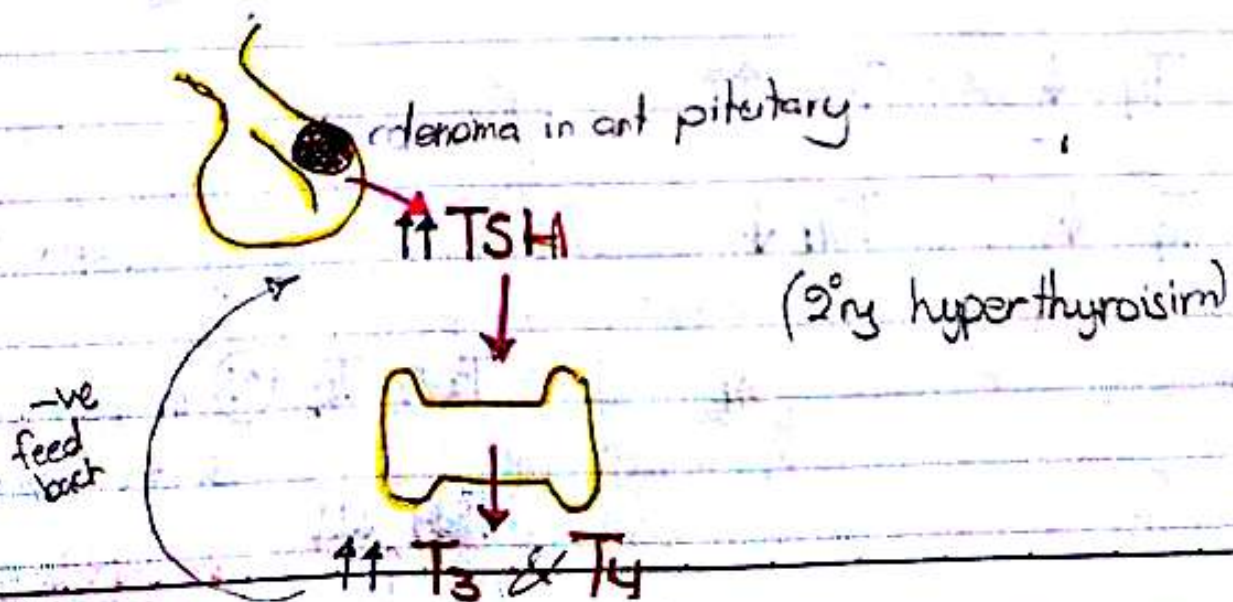
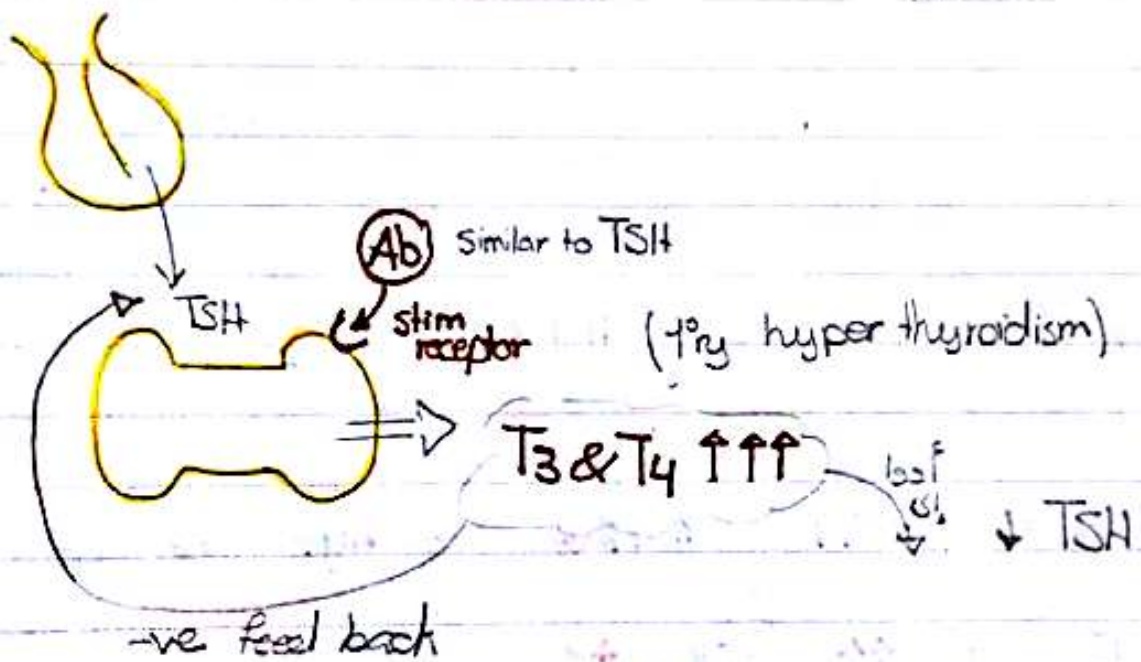
TSH

T₃T₄

7 hrs half life

14 days more accurate (long half life)

* Case 8 -

(T₃ & T₄) ↑↑↑ = Thyrotoxicosis:

⑤

$T_3 \& T_4 \uparrow\uparrow$

$TSH \uparrow\uparrow$



$\uparrow\uparrow TSH$

$\uparrow\uparrow\uparrow T_3 \& T_4$

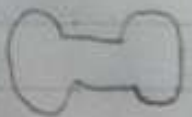
Localized Thyroid Test
function test
(best parameter)

Pituitary ← السبب
Thyroid ← مختلفات

$\downarrow\downarrow T_3 \& T_4 \rightarrow \uparrow\uparrow\uparrow TSH$

$\downarrow TSH \rightarrow \downarrow\downarrow T_3 \& T_4$

$T_3 \& T_4 \uparrow\uparrow$ $TSH \downarrow$ → 1° hypothyroidism



$T_3 \& T_4 \uparrow\uparrow$ $TSH \uparrow\uparrow$ 2° hyper



$T_3 \& T_4 \downarrow$ $TSH \uparrow\uparrow$ 1° hypo

$T_3 \& T_4 \downarrow$ $TSH \downarrow$ 2°

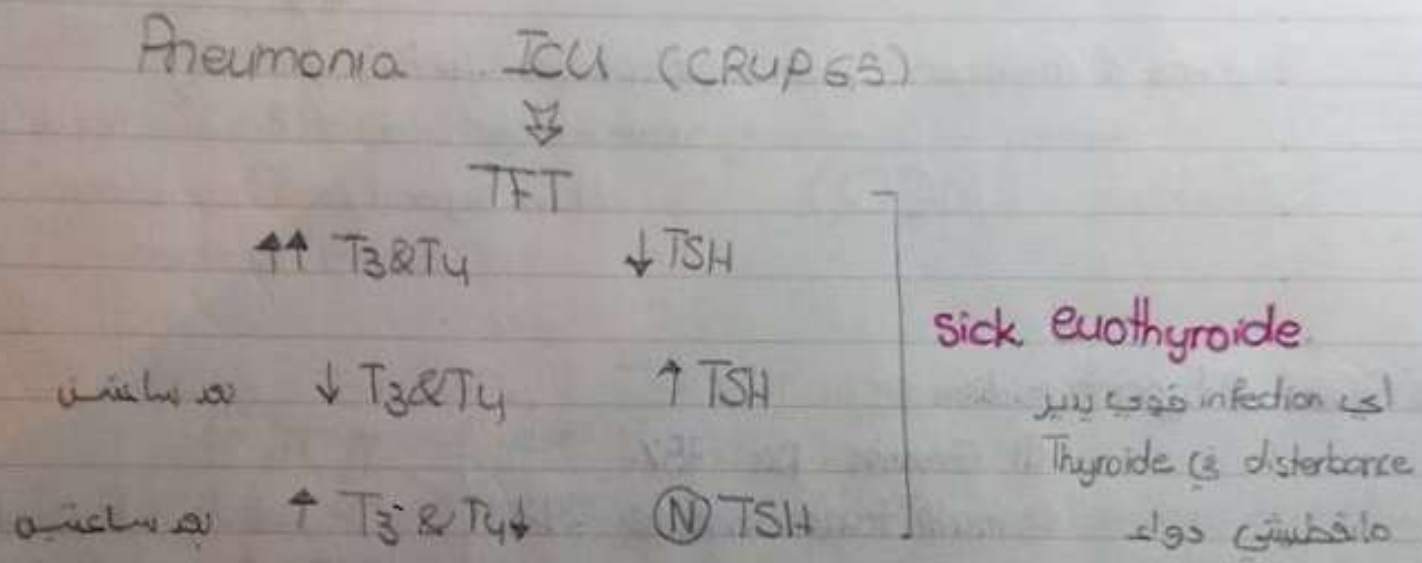
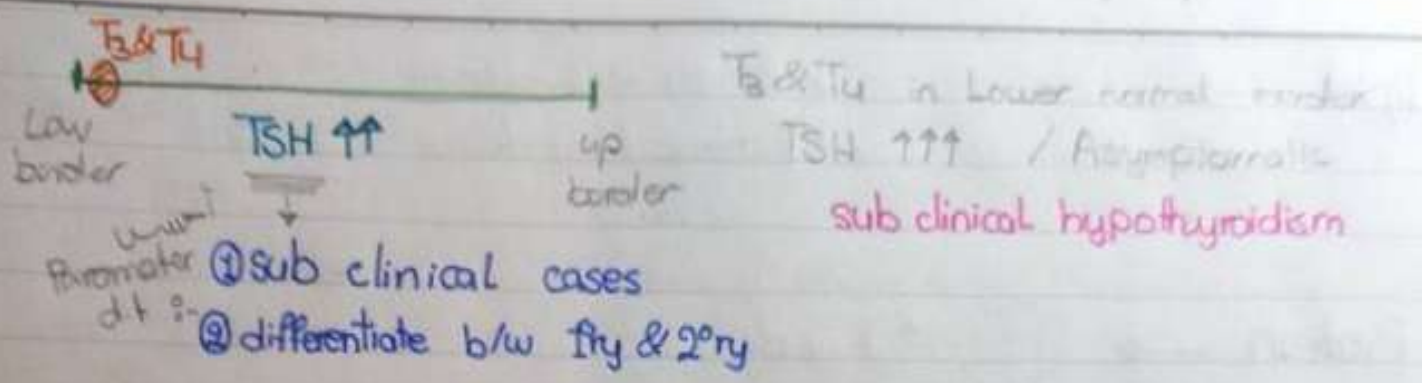


$T_3 \& T_4$ (N) in upper border
& Pte asymptomatic

$TSH \downarrow\downarrow$

subclinical
hyperthyroidism
cases

⑥



* Thyroid hormone action :-

↑ basal metabolism CHO
lipid protein → energy + heat

lipid ↓ catecholamine → ↑ sensitive of receptor

Pheochromocytoma also + sympathetic overactivity d.t ↑ catecholamine
do urine analysis if ↑ catecholamine if (N) thyrotoxicosis

CHO → ↑ glucose ① ↑ glycogenolysis destruct glycogen
② glycogenesis amino acid → glucose
③ Thyroxine work as anti insulin → ↑ glucose

④ ↑ intestinal absorption of glucose
- glycolysis (2ry Diabetes)

⑦

lipid $\rightarrow$ $\downarrow$ cholesterol $\rightarrow$ $\downarrow$ atheroma & $\downarrow$ ischemia
in hypothyroidism pts $\rightarrow$ $\uparrow$ Ischemic heart disease

Protein $\rightarrow$  $\uparrow$ Thyroid
(osteoporosis)

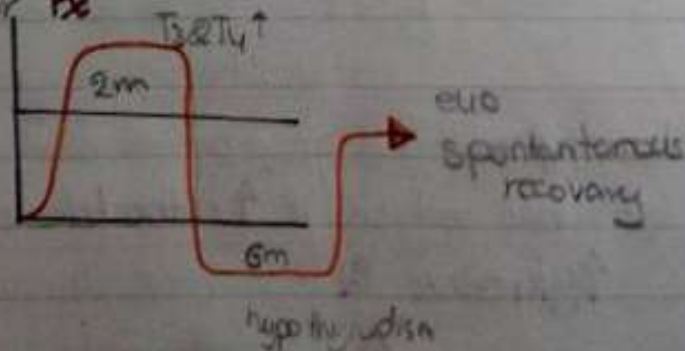
Thyroxine & Androgen $\rightarrow$ stim erythropoiesis $\rightarrow$ Polycythaemia $\uparrow$ Thymide
but Polymr erythema & warm hands is
✓ feature (MCQ) $\rightarrow$ hyperdynamic circulation

* causes of hyperthyroidism :-

- ① Graves Dis 75% } $T_3, T_4 \uparrow$
② multi toxic nodule 14% } $TSH \downarrow$ RAIU
③ single toxic nodule 5% } $RAIU \uparrow$ high only
- ④ Thyroiditis } Post viral Deg. (radioactive iodine uptake) 123
(ESR $\uparrow$) } Post partum
⊕ Painful neck (tenderness)

RAIU I 123 for Dx
131 for Rx

in Thyroiditis



Sick euthyroidism & Thyroiditis } → علاج
حول
نصير عليهم

فَمَرَّةً مَرَاتٍ يَنْحَوِي بِرُوحِهِمْ

- let fly for observation for 3 months

⑧

Graves
carbimazole

multiple

D.O.C

radiotherapy

rare < 1%

toxic nodule

single

D.O.C

radiotherapy definitive Rx / Propranolol Rx symptoms

⑤ exogenous, stroma ovary

⑥ ↑↑↑ TSH → pituitary Adenoma

↑↑↑ βHCG - hyper emesis gravidarum or cancer
↳ TSH like action

⑦ contrast media (IV)

⑧ drugs Amiodarone Type I, II hyperthyroidism
(Paradoxical action)

Atrial (systemic HTN / MS / Thyrotoxicosis) Rx: (β blocker / Ca channel blocker / Digoxin)
fibrillation vent Tachycardia D.O.C → Amiodarone

Don't stop even if Pte enter
in thyrotoxicosis.

[1]

epidemiology

Gravies Disease

most common cause of ↑ T₃ & T₄ (75%)

Autoimmune disease

HLA DR3, 2, BQ8 BQ9

(♀ > ♂)
9:1

↑ Smoking
↑ Infection

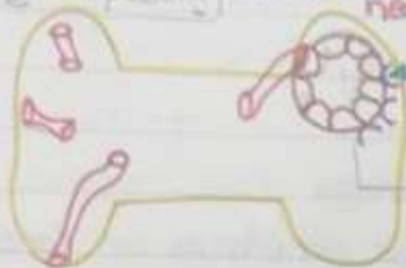
E. coli
Yersinia

Also other autoimmune especially

DM addition, vertigo, pernicious anemia

Investigation (TFT):-

- ↑↑ T₃ & T₄
- ↓↓ TSH
- Ab +ve 95% of cases



neovascular

TSH ↓

TSH receptor

Anti TSH R Ab → 95%

if not found doesn't

exclude

- Radioactive iodine uptake

↓
diffuse

Bruit over diffuse goiter = Graves disease

* Pathognomonic & [1] Bruit

[2] eye manifestation

worsening by smoking & radiotherapy

GAGs retro orbital



Protrusion then exophthalmos

Proptosis mid form of protrusion of eye ball, exophthalmos

sever form

(severity) ↑ up sclera

He Jial - up sclera of exophthalmos

walls of eye but not sclera



both eye involved but

asymmetrical

GAGs precipitate retrobulbar even before hyperthyroidism

Exophthalmos

ophthalmoplegia

GAGs in extraocular ms oblique anterior type 1st ms affected

red eye (conjunctival congestion ± edema (chemosis))

Optic Atrophy

(late stage)

→ dt congestion BVs & Dryness

→ sclera & sclera

[3] Peritibial myxedema

venules & BVs log Swiss Protrusion

non fitting

edema & red brown scaly skin

Precipitate GAGs sub cutaneous around tibia

التي

4 clubbing → Acropathy
+ tenderness in distal (radius)

Treatment

medical

surgery

Radiotherapy

<40 yr

>40 yr old age

Small goiter

huge cosmetically / pressure effect / retrosternal large goiter

(12-18 months)

① Carbimazole

Same for 4 weeks

then after month

40mg for 6 wks

5-20mg for rest of yr

OR

② Propyl thiouracil

the effect of Rx after 7-14 Days not follow up before 14 days

Follow up clinical improvement

CBC infection & sore throat

(stop drug) ✖ significant neutropenia (Agranulocytosis) leukopenia

S/E especially @ carbimazole

(2%)

Skin rash most common S/E

Propranolol may give to Rx symptoms

Propyl thiouracil D.O.C in pregnancy fat soluble drug cross placenta & Rx even hypothyroidism even in baby & the pregnant ♀ cause Ab of Graves is IgG which can cross placenta

& in breast feeding D.O.C propyl thiouracil can't cross to milk

* **Surgery** : 80% successful rate / 15% enter in hypothyroidism / 5% resistance stay hyper → remove lobe or sub total thyroidectomy

not applicable in singer's or voice depended career

Disadvantage ① vocal cord palsy ② suffocation & bleeding ③ hypoparathyroidism

hypocalcemia → discontinuous

→ hypoparathyroidism

reimplant in Pircan for sub clavicle

if medical failed

3

DCC medical failed surgery failed radiotherapy

* **Radiotherapy** - C/I in pregnancy & breast feeding

→ in fit not possible

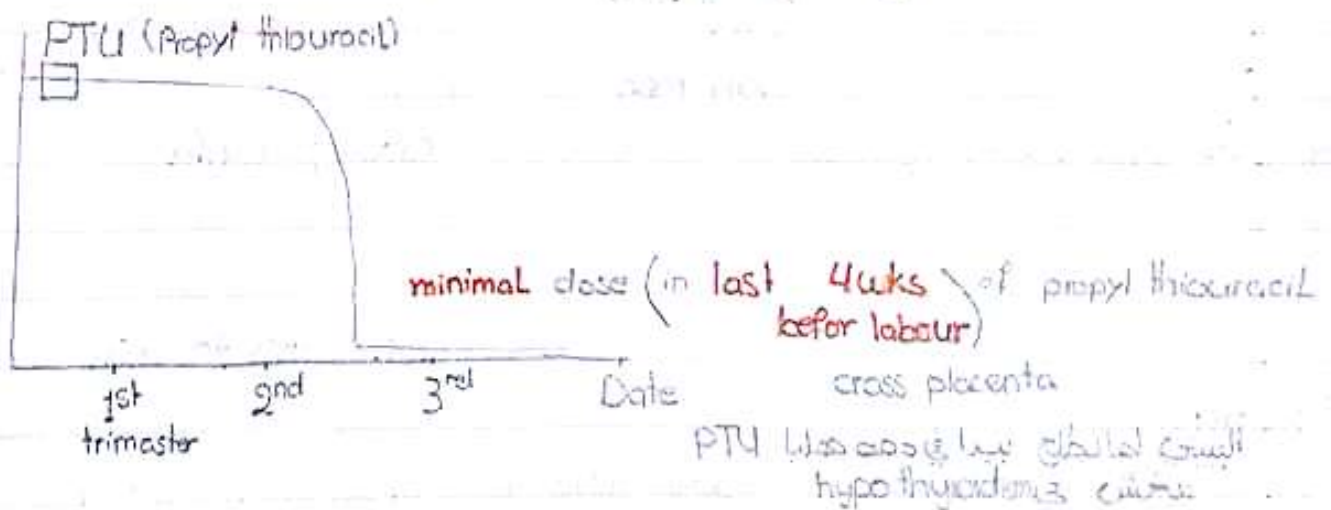
50% over 65 enter hypothyroidism

before surgery ↓ Thyroid hormones to avoid crises

give transient drug not therapeutic just ↓ Thyroid H Pre-operative

Propionolol + Na iodate (↓ hormones during 24 hr)

Thyrotoxicosis in pregnancy



* failed to R:-

- not respond from thy treatment
- relapse after respond

recurrent rate 50%

if functional best $\rightarrow$ stay benign not
convert to malignancy

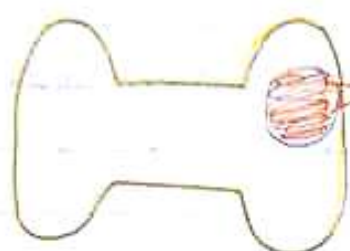
Toxic nodules

multiple $\longleftrightarrow$ single

unknown cause (idiopathic, Genetic)

sym & sign of hyperthyroidism

Pathognomonic eye manifestation of Graves
mainly affected on heart $\rightarrow$ AF esp if function
sweating / wt loss / diarrhea / palpitation



$T_3 \& T_4 \uparrow$

TSH $\downarrow$

Ab -ve

RAIU Localized to nodule area.

carbimazole & thiouracil have No role

Px:- Radiotherapy Rx of choice

Surgical

propranolol or Nadolol for symptoms only

& till the main line of Rx available

* Sub clinical cases:-

① TSH $\uparrow$ or $\downarrow$ ② $T_3 \& T_4$ (N) « free T_4 better »

Thyrotoxic crises (severe form of hyperthyroidism)

* ① infection destruction

* ② not prepared well to surgery

③ any manipulation « in bed side »

④ exogenous

C/P:- Hyperthermia temperature $> 42^{\circ}$
convulsion

coma

HTN

cardiac arrest & death

- Mx:-
- ① Cooling IVF rapid infusion
 - ② O₂, Antibiotics
 - ③ carbamazepine or Propyl thiouracil
 - ④ Propranolol ^{or} Ca²⁺ channel blocker to avoid cardiac arrest
(Anti-sympathetic)
 - ⑤ Analgesia $\rightarrow$ $\downarrow$ temp any type except aspirin d.t $\uparrow$ crisis
 - ⑥ if still hypoventilation Na⁺ ipodate may used
- # Rare but 10% of pte die

Hypothyroidism

* causes:-

① Auto immune

Hashimoto's thyroiditis (most common)

Atrophic thyroiditis

over dose - ② Post surgical resection & replacement

③ Post treatment post radiotherapy

④ Lithium, Amiodarone

⑤ some cases of Graves under effect of Ab $< 10\%$ inhibitory

* Hashimoto's:-

Ab against (Thyroid peroxidase) destruct follicular

$\uparrow \uparrow$ Thyroglobin ($\uparrow$ T₃ & T₄ release)

Lithium

• hypothyroidism

• hyperparathyroidism

• abnormal renal function



Ab against Thyroid peroxidase / Ab against Thyroglobulin
hyperthyroidism in early stage & transient (alt destruction of follicles) then euthyroid then persistent hypothyroid

↑↑↑ T_3 & T_4

Smoking ↓ risk of Hashimoto's

Hashimoto's ↑ risk Thyroid Lymphoma (mae) Precancer

Sjogren's (↑ risk to lymphoma)

* Investigation :-

TFT

T_3 & T_4 ↓

TSH ↑

Ab +ve Anti thyroid peroxidase (Anti-TSH Ab may +ve)

RIU

diffuse goiter

decrease uptake

↳ mostly stimulatory but

10% of cases inhibitory

in ECG

Low voltage ECG

(QRS in lead I, II, III) ↳ Pleural effusion

Pericardial effusion

Amyloid deposition

hypothyroidism

* Treatment :- start as low dose then ↑ dose
give Hormone «Thyroxin therapy»

Follow up by DEXA scan to Dx osteoporosis

«Dual energy X-Ray absorptiometry»

adjustment of thyroid replacement according to the case

HTN or IHD ~~~~~ ↓ dose

Pregnancy ~~~~~ ↑ dose L-thyroxin



* case 8 -

Hypothyroidism & HTN exclude

Low dose L-thyroxine (Low dose L-thyroxine) دوز کم لیتروکسین

Pan hypopituitarism تشخیص کلیه غدد پیتوئتری

TSH و GH و PRL و ACTH و Cortisol و Testosterone

L-Thyroxine کسر لیتروکسین

↑ metabolism
severe adrenal crisis

↓ GH ↓ ACTH then ↓ TSH

- No Cortisone
- No Androgen

Pan hypopituitarism

Never give L-thyroxine before exclude (Adrenal insufficiency) → give hydrocortisone IV

* Myxedematous coma *

↓ vital sign

hypothermia warming

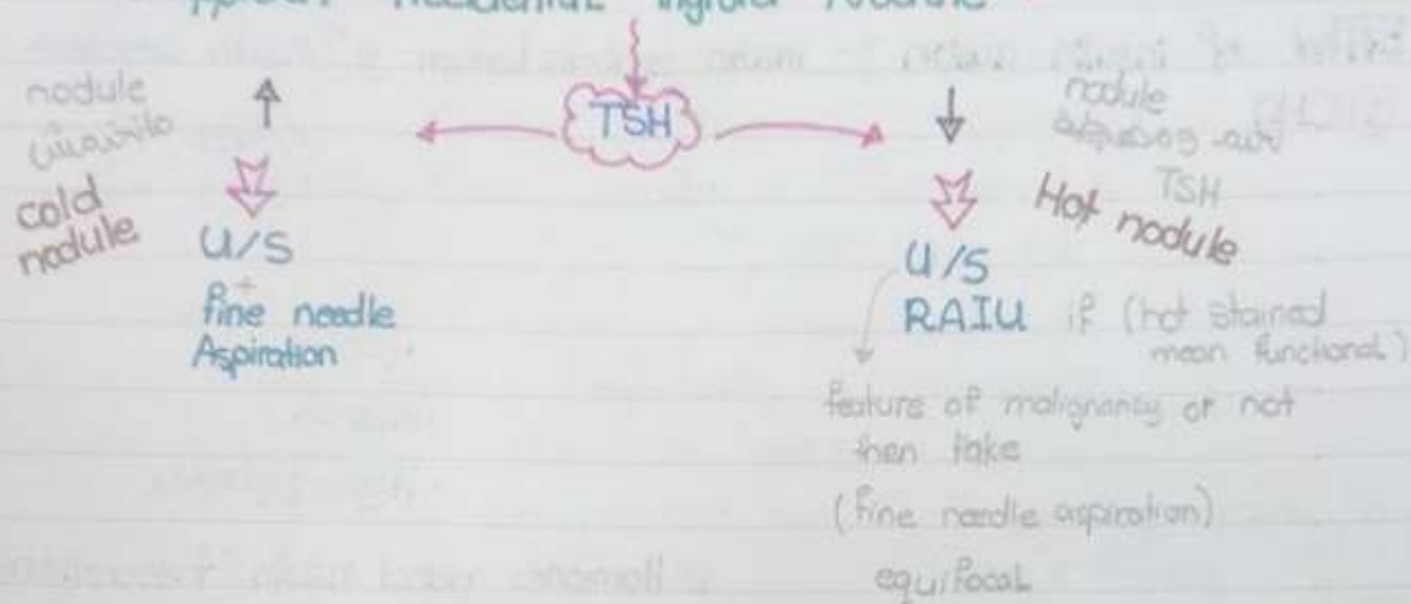
hypotension IV fluid

hypoventilation

Bradycardia O₂

Rx:- cortisone then L-thyroxine

* Approach Accidental thyroid Nodule *



DM (diabetes mellitus)

* Insuline 8-14

secreted by β -cell of pancreas

Hypoglycemia after Mx

check c-peptide in the Pte

CT scan on abdomen

↑↑ inside blood
insulinoma (usually)
sulfenuria toxicity

↓ in blood
exogenous
 Insuline
 (بنتجدر)

urine
 و متوقع لو فيه
 Sulfenuria product

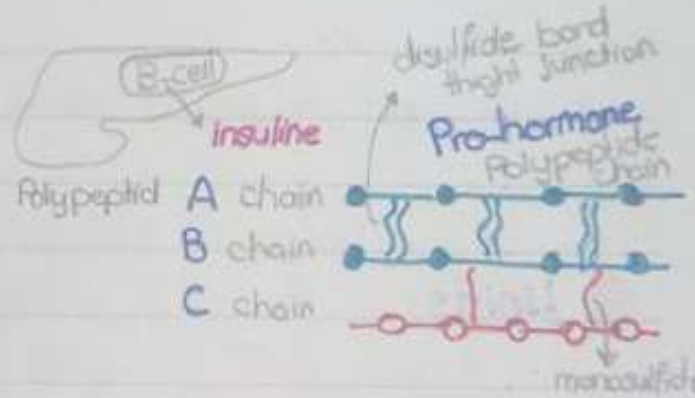
* Case 8-

حالة 8- hypoglycemia و أمها عندها سكر نتجالح بي hypoglycemia
 واحدة ادوية سكر بس تنتجر (تقدر تحم 1wk)

Pte should be under observation at least 48-72 hr

methformin not cause hypoglycemia ① ↓ glucose by stop liver
 sulfenuria stim B cell to secret insulin ② ↑ sensitivity of receptor to insulin

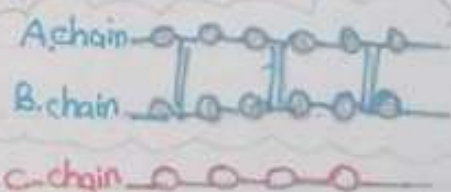
inactive سلفيد



C-peptide

A chain
 B chain
 active

active سلفيد secretion تنتج



* Effect of insulin action :- insulin anabolic hormone # ^{stimulatory} ↑ insulin secretion

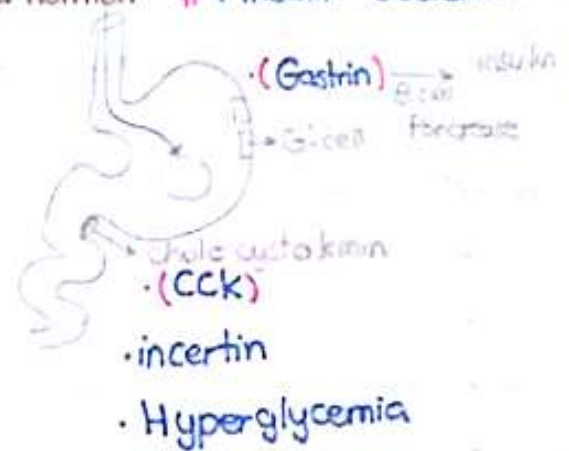
① CHO

↓ glucose :-

↑ entrance of G cell

↓ glycogenolysis

↓ gluconeogenesis



Hormones against insulin (↓ secretion)

causes of
type 2 diabetes

Thyroxine

GH. Acromegaly

Cortisone Cushing

Catecholamine (Adrenaline)

glucagon
glucagonoma

② Lipids

$\xrightarrow[\text{in liver}]{\beta\text{-oxidase}}$

Free fatty acids

β hydroxy Acetic acid

ketone bodies

insuline (-ve lipids metabolism) lipodystrophy + wt gain
(-ve protein metabolism) no free amino acids

DKA ~ type I DM
Ab attack all β cell (no insulin)

ket resistance type II DM

• receptor of insulin resistance

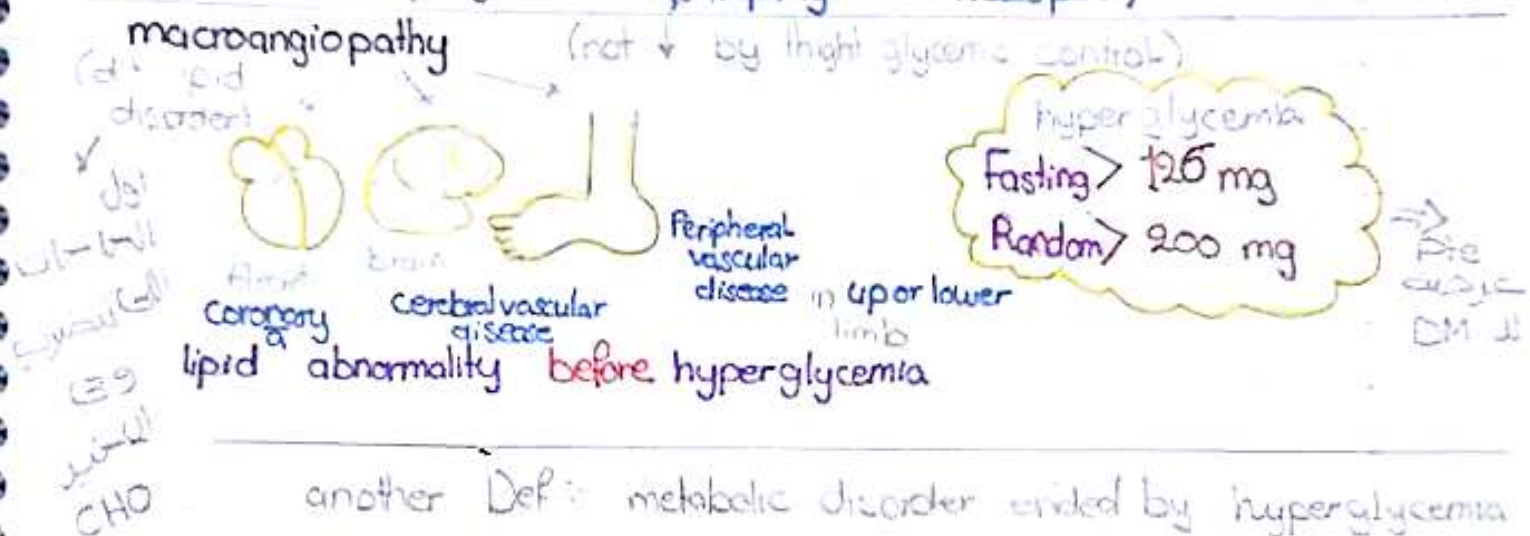
• β cell secret (few insulin)

lipodystrophy

↳ if inject insulin at some site

* Insuline S/E :- hypoglycemia
wt gain

* Def of DM: clinical syndrome of metabolic disorder characterized by hyperglycemia that sufficient to cause microangiopathy caused absolute insulin ^{type I} def or insulin resistance.



another Def: metabolic disorder ended by hyperglycemia

lipid abnormality happen before hyperglycemia

macroangiopathy not ↓ by tight glycemic control

↓ by aspirin → IHD

clopidogrel → CVA

statin & exercise

microangiopathy delay by tight glycemic control

most common cause of leg amputation & ch. renal failure

→ DM

DM type 2 more common 85%
type I more in ♂ / type II ♀ = ♀

* Acute complication of DM:-

- ① DKA
- ② Hyperosmolar non ketotic state
- ③ lactic acidosis
- ④ hypoglycemia

Endocrine (4)

1

FBS >126 gram & RBS >200 gram to cause macrovascular complication
macrovascular complication happen before microvascular complication
once u Dx DM(I), screening test for complication after 5 yr / Type II at same time of Dx

DM(I)

DM(II)

epidemiology

Absolute insulin def / glucose in urine >180
onset teen age group <40 yr
sever hyperglycemia
Renal threshold 180
O → > ♀

resistance in receptor / few auto antibodies
transient hyperglycemia / >40 yr

♂ = ♀

more in white occasion

more in black

Genetic

both Parents
50% pte will develop DM

both Parents
100% pte will develop DM

Genetic
susceptible
more

Twin (50% other twins
DM)

Twin DM other twin develop 90%

mechanism

Autoimmune - Ab against
B-cell (90%)

No Ab circulating

Amyloid tissue precipitated

C/P

Glucose can't be
satisfy center
Polyphagia / Polydipsia / Polyuria
osmotic sugar diuresis
thin wt loss (lipolysis not inhibited)

early presented & Dx

wt gain
insulin deficiency
-ve to lipolysis

Acanthosis nigricans
roughness rigidity

DKA (no insulin)

↑ lipolysis → ↑ keton body

Hyperosmolar Hyperglycemic non

ketotic state (more danger

, more fatal) keto resistance → keton body
can't be product in minimal of insulin

life style 3-6 months

Treatment

life style modification

+ insulin

if failed oral hypoglycemic

agent

1st always D.O.C after change life style
metformin

- ② TAG > 150 mg
- ③ HDL < 40
- ④ FBS = 110
- ⑤ BP $\geq \frac{130}{80}$

* Mx:- to avoid (DM, HTN, Ischemia)

- . Statin
- . Aspirin
- . ACE I
- . wt: reduction

Polycystic ovarian \$:- ↑ resistance to insulin
(Pre-diabetic) type II mainly Mx by **metformin**
metformin ↓ wt & cut ↓ risk of Diabetes

chronic * C/P of DM *

- ① Polyuria 2-3L / day
- ② Polydipsia
- ③ nocturnal uria
- ④ Polyphagia

DKA 1st presenting symptoms No insulin, ketone body
blood acidic (irritation sever gastric pain + vomiting & diarrhea)
Polyuria

Pte **Dehydration** (most cause of Death) so 1st line of Mx → **rehydration**

② rapid deep breathing (Kussmaul breath) type of acidosis (high anion gap)

Infection genital infection in ♀ & ♂
(d.t. ↓ immunity)
recurrent & delay healing
recurrent valvitis (♀) / Perinitis
recurrent UTI

Teeth loss d.t. collagen abnormality

macrovascular complication في مخرج الشرايين (screening type II) 4

Generalized Pruritis Sugar 180 - 200 mg/dL

in 2017 no more recommended screening test to Dx DM (time & cost consuming)

* Dx of DM :- normal

- FBS < 110

- Random < 140

impaired

> 110 < 126

> 140 < 200

DM

> 126

> 200



oral glucose tolerance test OGTT

صيام في حب (8 hr)

Glucose 75 نقطة
والتقسيم بعد ساعتين

FBS < 110 = N

RBS < 140 = N

> 126 = DM

> 200 = DM

> 110 < 126

> 140 < 200

Dx impaired DM
Prediabetic

impaired glucose (↑ risk for macrovascular tolerance complication)

(one reading + symptomatic pte) enough to Dx DM 2 reading if asymptomatic (test again)

HbA1C not used for DX used in "follow up"

d.t depend on other parameters

glyated Hemoglobin :- each 3 months HbA1C

vein section & dialyzer (falsly Low)

microvascular complication related to HbA1C

macro " " " " OGTT

Fructose amine :- (sugar + Albumin)

2-3wk state of glucose inside blood

normal ratio (6% only attached to RBC).

Avoid diabetes :- Dx pts w/ DM & on sugar test (hyperglycemic Dx)

Potential diabetes :- Asymptomatic, investigation (S), +ve family Hx

Urine analysis :-

① **glucoseuria**. (^{glucose} >180mg) searched in urine **not specific or sensitive**
not used for (Dx, follow up, indicate complication)

[Thyrotoxicosis / Partial gastrectomy / renal failure / pregnancy]

→ have glucosuria not in follow up (dt old age people threshold of kidney ↑)

② **ketoneuria** used DM I, II → failure in B-cell

③ **microalbuminuria**

30-299 μ g microgram 1st earliest sign that detect start of renal failure
in follow up should do (ophthalmoscope & microalbuminuria) to detect retinopathy & nephropathy & both can Mx if Dx early

* ACE Inhibitor :-

DOC → DM, HTN

→ DM Renal failure
→ HTN Renal failure

signs of hypoglycemia (palpitation/tremor/sweating)

6

* Mx of DM* in type I & II

life style modification (medical nutrition & activity Therapy) MNT

① wt reduction (by exercise) (30 min/D) 4 D/ per week

② Diet CHO 50% / lipid 30% / Protein 20% / Fruit

③ Alcohol ^{sbp} complex leading to hypoglycemia & masking hypoglycemia

Symptoms

DM (I)

life style

insuline depending type

Rapid acting Lispro, Aspart (1/2 - 1 hr)

short acting (2 hr)

intermediate acting ^{نابوية} NPH (neutral PH) (isophane)

neutral protamin
Hydrogen

Long acting . Ultralente (nature)

. glargen (synthetic) constant 24hr & peak

Regim of insulines:- multiple drug

mixed (mixtard) (2/3 dose / 1/3 dose)

1/2 unit for 1st 50 kg

1 unit for rest of wt.

Short acting ^{بوجع} 2hr after break fast

Intermediate ^{على باقي اليوم}

Case:- Diabetic take (2/3) & (1/3) insulin mixtard

on morning found glucose level 270 gr fasting

↓ intermediate dose of night
not work

Mx → night intermediate dose ^(النهار)

multiple drug regimen $\rightarrow$ tight glycemic control

miscommon cause of Diabetic pt $\bar{E}$ hypoglycemia $\rightarrow$ missed meal

diabetic pt ask to take frequent meal

Ideal ① $HbA_{1C} < 7$
in diabetic

② FBS	90 - 130	accepted 160
③ RBC	180	accepted ≤ 200

* case :-

70yr Diabetic take insulin check glucose level found hypoglycemia
recurrent hypoglycemic attack

Mx :- $\downarrow$ dose of insulin

Tight glycemic control is not

HbA_{1C} $\downarrow$ Target measurement is not

Autonomic nervous system
masking of hypoglycemia
Neuropathy
Symptoms

* Oral :-

insulin injection S/c, IV infusion pump in hospital IM in case of
venous collapse of DKA pt.

No Oral insulin

Site of injection :- Abdomen (best site)

thigh

Arms

- after injection do massage (warming ↑ distribution of insulin)

- change site of injection

- inject perpendicular (قصورقش - مؤصلا لالم)

- saving at fridge

lymphatic infection
subcutaneous fat area
Alcohol
ما تحطش قباله
قبل ما تحزبه
خلع الالسنولن
وخليه يرفق شوبه

* Endocrine (5) :-

* Mx DM(II) :-

- ① life style modification (3-6 months)
 - ② Tablets (in some cases after doing screening & macroalbuminuria start tablet immediate) screening of type II at time of Dx
 - ③ Morbid obesity BMI ≥ 35
 - ④ Retinopathy
 - ⑤ Nephropathy
 - ⑥ HbA_{1c} ≥ 9
- start on tablet
don't do life style modification

Oral hypoglycemic agent :-

- A** Sulphonylurea - Gliclazide / Glibenclamide / Tolbutamide
Chlorpropamide (D.O.C in thin people)
- B** Meglitinides - Repaglinide / Nateglinide
↓ (hepatotoxic)
- M.O.A ↑ insulin release from B-cell from pancreas
- less one to cause hypoglycemia
Short acting drugs
- S/E
wt gain & hypoglycemia

- C** Biguanides Metformin (always start 1st)
(D.O.C in obese people)

- D** Thiazolidinediones Rosiglitazone / Pioglitazone / Thio glitazone

M.O.A ↓ resistance / ↓ gluconeogenesis (liver glucose output)

* Side effect of insulin :-

- ① Hypoglycemia
- ② wt gain / Allergy / insulin resistance
- ③ lipodystrophy at injection site

Lipoma of fat is sub cutaneous

site of insulin injection
lipodystrophy

Sever hypoglycemia → insulin → warming → Hypoglycemia

Pte & DM (II) & have recurrent attack of hypoglycemia
avoid (sulphonylurea / Meglitinides)

②

other use of insulin:- ① Rx of hyperkalemia ② stimulatory test of GH
③ insulin tolerance test in panhypopituitarism

→ avoid sulphonylurea in elderly → may have autoneuropathy pte can't feel

* **Methformin action:** ^{hypoglycemic symptoms}

① ↓ gluconeogenesis

② ↓ glucose Absorption (intestine) → Malabsorption Gas & bloating

③ ↑ sensitivity insulin receptor

S/E:- GIT upset / vit B12 absorption

Diarrhea

degrade in liver to lactic acid & excreted in kidney

avoid giving Biguanides (Methformin)

C/I Failure of heart, renal, liver (↓ metabolism)

99.9% not cause hypoglycemia 0.1% can cause in diabetic pte

- Contrast pte

IVU

(البديل للـ sulphonyl urea ← methformin)

④ kidney الـ kidney

4B Metformin

contrast

M.O.A :- ↑ receptor sensitivity

except Metformin coabli agent ^{oral hypoglycemic}

Metformin + Acarbose → C/I both have same S/E

Diarrhea + bloating

E Acrobase (α glucosidase inhibitor) يعتبر من مثبطات إنزيم الجلوكوزيداز
 Diarrhea & bloating (↓ absorption of glucose) intestinal

Sulphonylurea Part of it free (active)
 another part binding to albumin & carrier serum.

Don't mix it $\bar{e}$ Aspirin & antifungal • prolonged action of sulphonylurea

Alc $\bar{e}$ free all binding severe hypotension

insulin is usually mixed $\bar{e}$ **metformin** not causing hypoglycemia

any pte $\bar{e}$ CRF or renal disease you should $\downarrow$ dose of insulin

dt insulin secreted by kidney

case 8- 35y ♀ known case of DM 60unit RFT $\rightarrow$ creatinin **[3]**

recurrent attack tremor, sweating & palpitation HbA_{1c} $\bar{e}$ normal level

sugar **[60]**

creatinin 3 & Normal $\rightarrow$ 1 & Renal Failure

Mx is $[\downarrow \text{insulin dose}] \rightarrow$ dt insulin is secreted by kidney
 والحالة عندها $\leftarrow$ renal failure $\leftarrow$ insulin يفرز أبطأ

عندها يفرز أكثر

لو كانت نفس الحالة ولا creatinin **(N)** دكتور إن ال Pte ناخذ

2 drugs that $\downarrow$ glucose

case :- ♀ known case of DM (newly married) 4 months from marriage presented

$\bar{e}$ recurrent attack of hyperglycemia & acidosis on insulin 60unit

Mx:- Rx the DKA then $\uparrow$ insulin dose dt pregnancy placental lactogen

$\uparrow$ glucose $\rightarrow$ **Insulin** $\rightarrow$ $\uparrow$ dose in pregnancy, infection, infection, surgery

$\rightarrow$ $\downarrow$ dose in renal failure

Glucagon + glucose

* **exenatide** :- Injection S/C for type II DM

R (Type II)

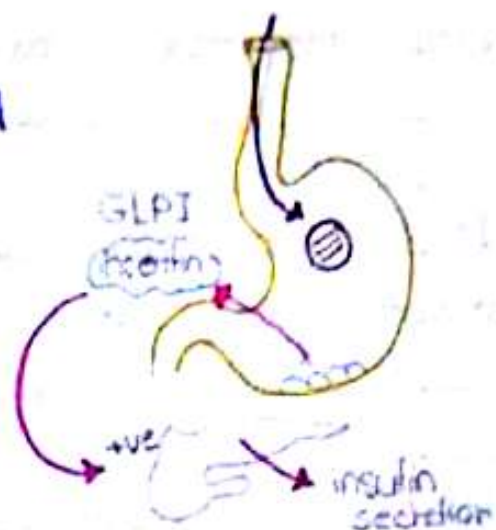
(No hypoglycemia)

incentine (Glucagon like peptide I)

+ve insulin secretion

↓ gastric empty

↓ appetite (↓ wt)



S/E :- pancreatitis

to Mix type II DM start @ 1 Drug (ما نستخدم اول ما نورد glucose ببطء)

not control → 2 drugs not control → triple tablet

therapy not control → & not reach high

glycemic control → switch to insulin injection (Hypoglycemia يدبر ببطء)

Target goal HbA1C < 7% / FBS 90-130 / RBS < 180

* **Special situation** :-

* **Honey moon period** :- Type I DM on **insulin** therapy & transient refresh of β -cell secret endogenous **insulin** → Pte enter in state of hypoglycemia

stay on same dose of insulin but ↑ **snacks of meals**

(very high dose)

* **Somogyi phenomena** :- take dose of night pte enter to hypoglycemia at

3a.m (the body to compensate secret stress hormone like thyroxine, cortisone, glucagone count regulatory hormone) ↑ glucose level

The pte when check morning found (state of) hyperglycemia

↑ **Dawn phenomena** :- evening dose low $\rightarrow$ Ate on morning hyperglycemia
dose not enough to cover the night increasing of glucose $\rightarrow$ **nightmare**

D/D of fasting morning hyperglycemia $\rightarrow$ Dawn $\rightarrow$ at 3am hyperglycemia
Smoggy $\rightarrow$ at 3am hypoglycemia
to differentiate * check glucose level after mid night at 3am measurement

* **case** :- 35yr known case of DM since 20yr back on insulin.
(adren disease) presented with hyper pigmented lips & depigmented patch on skin
(vitiligo)
pt c/o of recurrent attacks of hypoglycemia since 2 months
addison's $\downarrow$ cortisone & adrenal gland affected

if known case:
type I
addison's

if recently Dx
honey moon

* complication of DM *

Acute

chronic

micro

macro

Retinopathy

90%

treatable

Neuropathy

70%

Non-treatable

nephropathy

30%

treatable

screening by

ophthalmoscope

non-proliferative

Follow up

Proliferative $\rightarrow$ laser

need 20yr of DM

Dx

by microalbuminuria
if $\uparrow \rightarrow$ ACE I

oral med
in OSCE

DM HTN TB asthma

6

macroangiopathy not related to that glycaemic control. Non preventable.

- heart (coronary a) ① stat +
- brain (CVA) ② Aspirin 75 or 100 mg
- peripheral vascular ③ Statin supposed to be:-
 - TAG < 150
 - LDL 100
 - HDL > 40
 - BP < 130/80
- ④ ACE I

* DM (neuropathy) * a whole hand to give

sensory

motor

ANS

spinal cord

Deep
↓
charcot
joint
(1st affected
in sensory)

symmetrical
↓
gloves
stocks
sensory
loss bilateral

↓
asymmetrical
• Mononeuropathy
• mononeuritis multiplex
• poly neuropathy

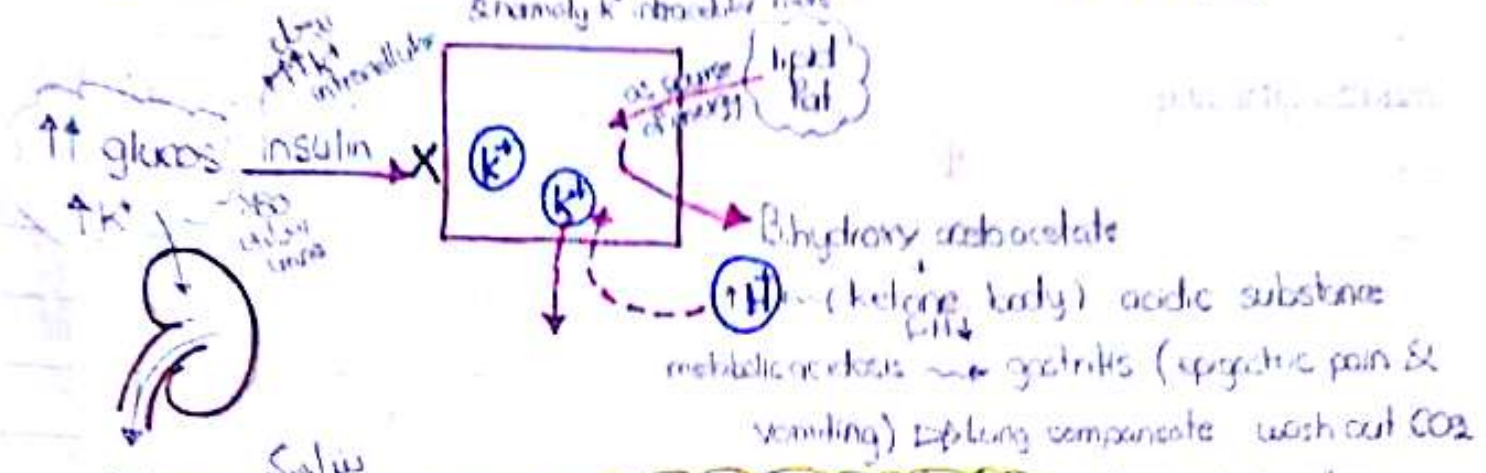
Diabetic
Amyotrophy
mixed lesion up &
lower
mus. atrophy
Burning skin

- ① postural hypotension
- ② +12 not increases after movement or standing
- ③ alternating bowel habit
- ④ Gastroparesis
- ⑤ masking hypoglycaemia
- aneemia
- Addison's
- Danties
- DM

الحساسية

DKA
 Diabetic keto acidosis (Present very early of DM I)
 Pain & severe vomiting
 & usually K^+ intracellular move

Hyperosmolar hyperglycemic non ketotic state
 in DM(II)



In DKA K^+ ↑ or (N) ↓
 ↓ insulin ↓ glucose ↓
 ↓ K^+ ↓

dehydration ① by vomiting ② polyuria ③ kussmaul breathing
 (main cause of death) 1st line of Mx (Rehydration)

* Osmolarity of blood calculate 8-

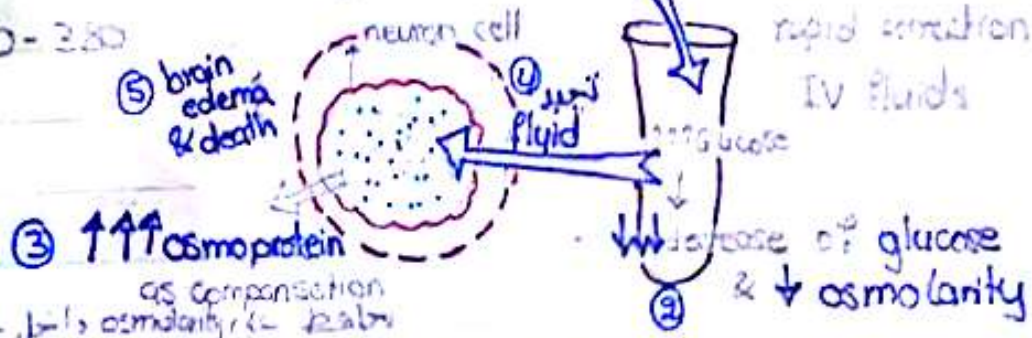
$2Na^+ + K^+ + \text{glucose} + \text{BUN}$ in DKA ↑ K^+ & ↑↑ glucose 400-500

osmolarity (N) = 275 - 295

DKA >300 - 320 osmolarity

HHS >340 - 380

IVF ① in & DKA of Dehydration rapid correction & over dose IV fluids



start of IV fluid normal saline 0.9 NaCl

when glucose 270mg change into Dextrose

reach

Saline 5%

mortality rate 10%

8

Brain edema in DKA :- ① rapid drop in glucose level
② Na bicarb (the only indication in severe acidosis
<7.1)

Hyperosmolar Hyperglycemic non ketotic state :-

in Type II DM . few insulin still secreted (No lipolysis)

↑↑↑↑ glucose



due to insulin cause inhibit lipolysis
(lipid destruction inhibition)

No keton body (ketoresistance)

No acidosis ~ No pain or vomiting
very late presenting

↑↑↑ glucose reach up to 1000-1500

high osmolarity more sever than DKA

>340 - 380

>300-320

high risk for thrombosis & DVT

mortality rate **40%** ① in early state asymptomatic

② die by thrombosis or hypoglycemia

* How to differentiate b/w DKA & HHS :-

① PH Normal (7.35-7.45)

② osmolarity (must be in mmol measurement to calculate) Normal <300

③ CO₂ (↓ in DKA) Normal (35-45)

osmolarity ↑ , PH ① , CO₂ ① → Hyperosmolar hyperglycemic non ketotic state.

* causes of DKA :-

- ① missed insulin dose
- ② infection (most common cause)
- ③ infarction
- ④ cocaine abuse (sympathetic)

* DKA vital sign :-

- ① Tachycardia (dehydration)
- ② fever
- ③ hypoten
- ④ $\uparrow$ Respiratory rate
- ⑤ WBC $\uparrow$ (Leukocytosis) ... stress Leukocytosis not feature of infection

To Dx DKA cause is infection ~ measure the **C-RP** if $\uparrow$ infection

Endocrine (6).

1

Coma end stage of DKA

most of cases presented awake C/O (polyuria, epigastric pain, vomiting)

Fever, Tachycardia, dehydration, Acetone smell

when measure glucose level 400-500

* DKA more common in IDDM (type I) *

* Symptoms :-

- ① polyuria polydipsia
- ② epigastric pain + vomiting (No diarrhea)
- ③ kussmaul breath
- ④ fever

* signs :- Dehydration Dry mucous membrane & skin
Acetone smell (mouth)
Tachycardia (weak pulse)

* Investigation :- Blood glucose 400-500
ketone body +++ (blood & urine)
ABG $\text{pH} = \downarrow \downarrow 7.3$
[$\text{CO}_2 < 35$ d.t kussmaul breath
Anion gap $\uparrow \uparrow > 18$ $[(\text{Na}^+ + \text{K}^+) - (\text{HCO}_3^- + \text{Cl}^-)]$
osmolality $\uparrow \uparrow 320$

* Rx :- **I** IVF by isotonic NaCl 0.9% or hypotonic 0.35

1 liter	30 min	} 6 Liters in 1 st 24 hr IVF 100 mL / kg
1 liter	1hr	
1 liter	2hr	
1 liter	4hr	
1 liter	8hr	

II correct electrolyte (K^+ 3.5 - 5.5)

if normal K^+ $\rightarrow$ 10 mmol KCl لا نريد insulin سكرنا لو سكرنا لو

Low K^+ $\rightarrow$ 20 mmol KCl rapid drop in K^+ سكرنا لو سكرنا لو

if K^+ $\uparrow$, don't give K^+

III Rx Acidemia if PH < 7.1 give $Na^+ HCO_3^-$ 1.4% IV.

IV Rx hyperglycemia Rapid acting Insulin infusion

50 unit in 50% dextrose saline

slow infusion
in ven

6 unit / hr

3 unit / hr when 270 glucose

2 unit / hr when 180 glucose

this concentrate
if given in small ven
will cause
Phlebitis
so give it in
central vein

if pts conscious start orpl feeding
(overlap b/w infusion & S/C insulin intermediate)

CO_2 in DKA wash out by hyper ventilation ($\downarrow CO_2$)

Na^+ normally 135 - 145 in DKA + Acute (rapid $\uparrow$ in glucose leading to $\downarrow$ in Na^+). BUN = $\frac{1}{2}$ urea

3

المقاييس لوزن سكريات (mmol/L)

mg → mmol

glucose
18

urea
24

Normally ↑ glucose ↓ Na⁺ to compensate
hyper osmolality 300-320

Anion gap = 18 cation +ve / anion -ve
Na⁺, K⁺ Cl⁻, HCO₃⁻
normal $[(Na + K) - (Cl + HCO_3)] = 12 - 18$

in metabolic acidosis $\begin{cases} \text{High anion gap} \\ \text{Normal anion gap} \end{cases}$

in DKA ↑ acids which contain (-ve charge) → ↑ anion
body as compensate يجب ان سبب خلل
as HCO₃

↑ HCO₃ → kidney ↓ any anion (which is Cl⁻)
one of D/D of high anion gap :- DKA
(> 18) methanol Alcohol
Ethylenglycol
Asprin salicylate

Metabolic acidosis due to High anion gap due to

osmolality ↑ → anion gap $\begin{cases} \uparrow \rightarrow \text{DKA} \\ \text{⊗} \rightarrow \text{HHS} \end{cases}$

in IV fluid should insert central line & urine catheter

to measure input output chart

if central pressure $\uparrow$ mean IV fluid rapidly effusion $\rightarrow \downarrow$ rate
 [N] & No urine out mean kidney not hydrated
 $\rightarrow \uparrow$ rate

urine output ① 35 mL/hr

when glucose level reach 270 switch to Dextrose saline 5%

فلس حذر Hyperglycemic

Insulin C/I in DKA if K^+ less than 3.5

* monitoring of DKA :-

- ① vital sign
- ② ECG $\rightarrow$ for K^+
- ③ check K^+ level
- ④ ABG

- may give heparin in prolonged bedridden or previous DVT

* Mx of HHS *

Some Rx as DKA except :-

- ① need less insulin
- ② need subcutaneous heparin Low molecular wt
- ③ IVF by hypotonic saline 0.35 NaCl

comatized pte known case of DM

type I give sugar

لو ما قدرتش تعطي IV أو oral

ما فيش حل إلا إنك تعطي IM glucagon

5

* Mx of hypoglycaemia *

- oral sugar if pte conscious or (IV Dextrose 5% or 20%)
- if pte not conscious give it IM glucagon

50% →
Prohibits

IM glucagon given

in case of

- B₂ blocker toxicity
- Hypoglycaemia

hypoglycaemia	DKA
Sympathetic overactivity	
Pupils dilated	
Tachycardia	Tachycardia
good volume pulse	low volume weak pulse
sweaty	acetone smell
cold hand	dehydrated pte
	fever

coma g DM Pte لو
sugar اعطى
hypoglycaemia لوفاق
لو. ل. عيش حديد فرق
كسر و يرفع السكر هلبا

* staging of thyroid gland :-

G₀ normal (not palpable not visible)

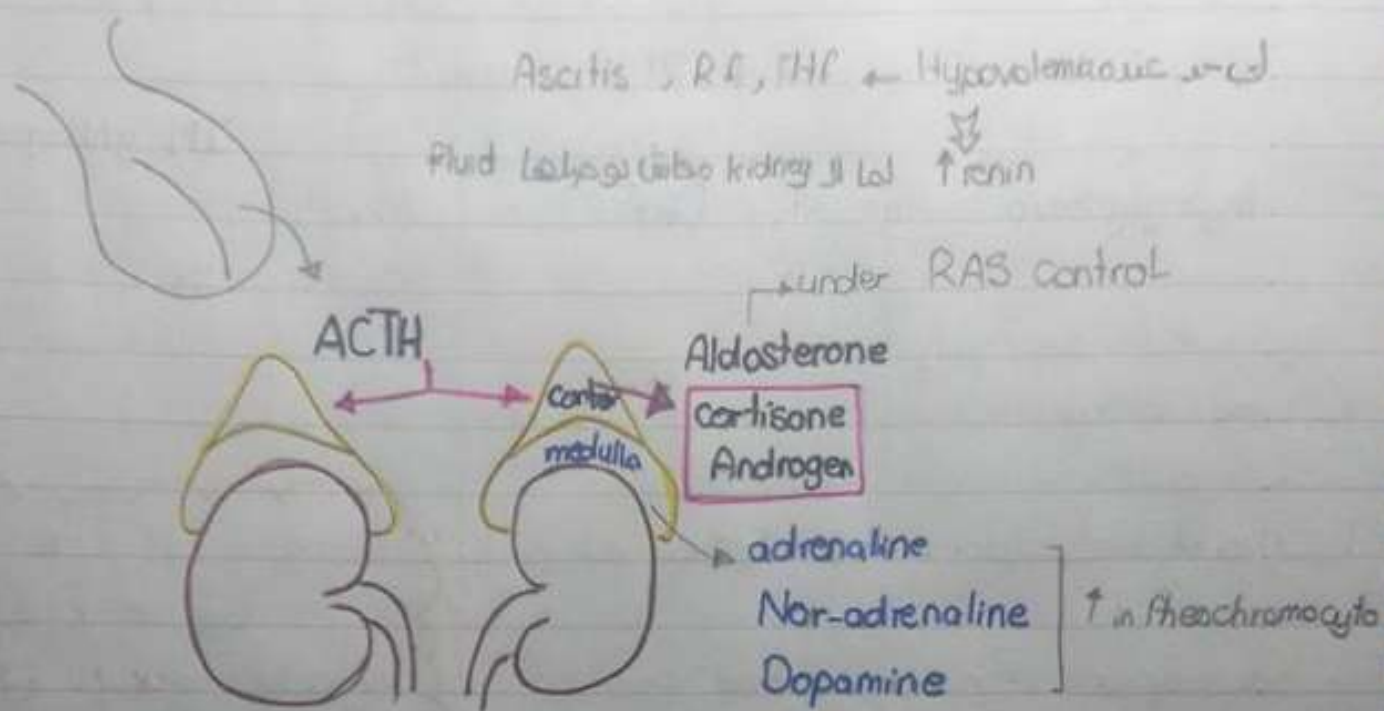
G₁ A not visible but palpable as terminal phalanges of th

B palpable & visible even when extended his neck

G₂ visible in neutral position

G₃ visible from a distance (ask pte to flex his neck if still appear visible Grade 3)

* Adrenal gland *



$\uparrow$ aldosterone $\rightarrow$ adenoma in cortex secrete aldosterone (Conn's $\$$)
 hyperaldosteronism

tumour $\uparrow$ cortisone (Cushing's $\$$)

Pte known case uncontrolled br. asthma presented w/ (moon face, central obesity, acne striae hirsutism)

feature of Cushing's d.t ch use of corticosteroid

Cushing's $\$$

* causes :-

- ① Iatrogenic (exogenous steroid)
- ② spontaneous Cushing's



ACTH depending causes

ACTH $\uparrow$ & Cortisone $\uparrow$

① Pituitary adenoma (Cushing's disease)

② Paraneoplastic $\$$ (lung cancer, carcinoid tumour)

ACTH independent causes

① Adenoma adrenal ACTH $\downarrow$ Cortisone $\uparrow$

② Hyperplasia

③ Carcinoma

♦ Treatment :- surgical is R of choice

Myrapone now days used in ↓ cortisone (Treat Cushing)

* Action of steroid (cortisone action) :-

① CHO ↑ glucose (↑ glycolysis, ↑ gluconeogenesis, ↓ insulin secretion)

FAT ↑ lipolysis (↓ sensitivity to catecholamine receptor)

Protein lysis → (Bone osteoporosis), skin (thin), B.Vs (Bruises)

② vit D (↓ Ca^{+2} & PO_4^- , inactive vit D)

③ Aldosterone like action stim action ↑ Na^+ H_2O (HTN & edema), ↓ K^+ ↑ H^+ metabolic acidosis

↓ K^+ ms Heart (arrhythmia), Sk ms (proximal weakness), smooth ms (constipation)

in Cushing
↑ cortisone

④ Androgen like action (acne, Hirsutism)

⑤ Bone marrow [↑ RBC (polycythemia), ↑ PMN, ↓ lymphocyte & eosinophile]

⑥ Anti allergic (↓ count of eosinophile) Anti inflammatory (↓ chemotaxis & phagocytosis & lysosomal activity)

⑦ Sexual (infertility)

⑧ Psychological disturbance

C/P:- DM, DI

Trunkal obesity / Moon face / Buffalo-hump

→ d.t catecholamine receptor more in periphery (↑ lipolysis thin periphery)

& central obesity, Lower lumbar back pain, thin skin, Bruises, acne

striae, osteomalacia, flushing, plethoric face, Hirsutism

Striae { Dark ($\uparrow$ ACTH) d.t stimulates melanocyte (melanocyte stim. Hormone)
Pink ($\downarrow$ ACTH)

* Investigation

I confirm cushing by

1st test

2nd test

Low dose dexamethasone

Suppression test 1mg

$\downarrow$ cortisol

(N)

Still $\uparrow$ cortisol

cushing &

or pseudocushing:-

Alcoholic
Depression
obesity

24 hr urine collection cortisol level

(N) ≤ 276 nmol

if >276 cushing

nelson &

Pituitary adenoma false localized
as adrenal adenoma & Mx by
bilateral adrenalectomy.

Pte presented &

dark hyperpigmented skin

& $\uparrow$ ACTH

Melanocyte

II Localization

measure ACTH level

if $\uparrow$ or (N)

dependant

independant

(Adrenal causes)

Pituitary

ectopic

Lung cancer
cardioid tumor

CT Abdomen

+ve
start R
Adrenalectomy

-ve

adrenal venous sample

if $\uparrow$ ACTH & cortisol

High dose dexamethasone (4mg)

suppression test

cortisol $\downarrow$

pituitary cause

still $\uparrow$ cortisol

ectopic

MRI / CXRay / CT scan

+ve

(N)

-ve

Trans-sphenoidal sample

Endocrine (7)

①

* Hyperaldosteronism *

1^{ry} Conn's \$
(↑PH, BP, Glucose)
systolic pressure >160

Causes:-

- ① Adrenoma
- ② Adrenal carcinoma

↑ aldosterone ↓ renin

C/P:- HTN; Odema (HTN in 2^{ry} only in renal & stenosis)

↓ K⁺ → muscle weakness, fatigue
palpitation 2^{ry} to arrhythmia
constipation

Hyperglycemia

polyuria

Metabolic alkalosis

Hypernatremia Hypokalemia

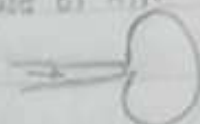
↓ (PH, BP, Glucose) adrenal insufficiency

Rx:- 1^{ry} surgery (hormone replacement)
2^{ry} spironolactone ± ACE I
HTN

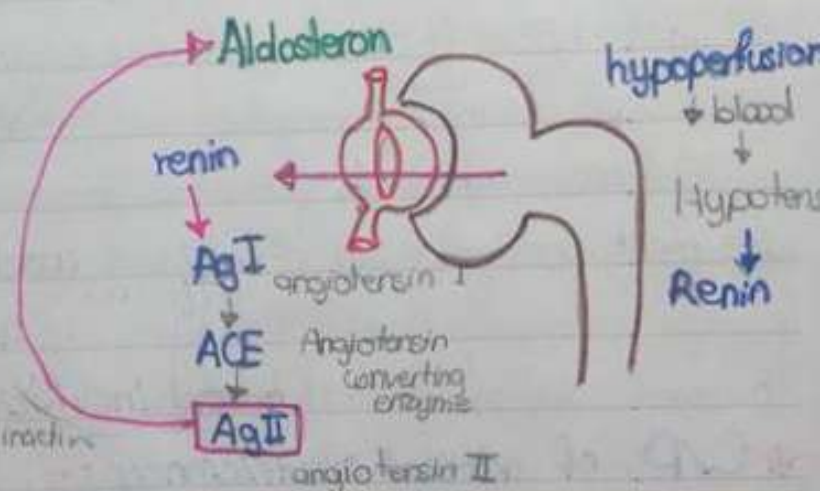
2^{ry} HTN

HTN

renal & stenosis
most common cause of HTN



↑ aldosterone ↑ renin



Heart failure → ↓ perfusion to organ (kidney) → RAS (Hypertension 2^{ry} to HF)

→ C/I in case of bilateral renal

& stenosis only spironolactone & f

& unilateral & stenosis ACEI can used

* Adrenal insufficiency *

↓ adrenal gland hormone

Addison's disease

1^{ry} (Adrenal destruction) $\downarrow$ hypoparathyroidism

Aldosterone (Hypotension, tinea)

Cortisone $\uparrow$ ACTH

Androgen pigmentation

Hyponatremia, hyperkalemia, metabolic acidosis

Causes:

- ① Auto immune $\leftarrow$ when $CD_4 < 200$
- ② Infection (TB, HIV, fungal) $\leftarrow$ MAC mycobacterium complex
- ③ waterhouse friedrich's (neisseria meningitidis)
- ④ Adrenalectomy $\leftarrow$ no replacement $\leftarrow$ pituitary gland disease
- ⑤ Adrenal metastasis (renal cell carcinoma, lung cancer, breast ca)

The most common cause of Addison's word wild $\rightarrow$ TB

" " " " " in united kingdom $\rightarrow$ Auto immune

The most common cause of adrenal insufficiency $\rightarrow$ steroid withdrawal

* C/P of adrenal insufficiency *

- ① $\downarrow$ aldosterone (Hypotension) $\leftarrow$ Postural hypotension
- ② Hyper pigmentation ($\uparrow$ ACTH) **only 1^{ry}**
lips, buccal, creases, Generalized hypertension
- ③ cortisone $\downarrow$ glucose weakness, fatigue
- ④ Androgen Hair loss, $\rightarrow$ Axillary &
- ⑤ Acidosis K^+ epigastric pain & vomiting

* hyper pigmented lips
• smoking
• PH polyp d
• Addison

Data: $\rightarrow$ acidosis $\leftarrow$ postural
Fatigue, epigastric pain, vomiting, hypotension, hyponatremia, hyperkalemia

• glucose $\leftarrow$ $\uparrow$ DKA

• K^+ & Na^+ $\leftarrow$ DKN $\leftarrow$ Addison $\leftarrow$ Na^+ $\uparrow$ K^+ $\downarrow$

PH in Addison & DKA

both acidosis

• Anion gap $\leftarrow$ DKA $\uparrow$ Addison $\downarrow$

③

* Investigation *

electrolyte

Na ↓
K ↑
PH ↓

only try

cortisone → ↓ glycogen
↑ Ca²⁺

Urea ↑ in
↓ in

Short sayan ACTH en

① * Sayan ACTH en test & Low dose

cortisone قبل و بعد ما يطيح ACTH (N) ← بي زيده

Dx Adrenal insufficiency
cortisone try
↓
2° try

↓ cortisone even after give ACTH

قيس
ACTH
الاولى قبل
sayan

↑ ACTH
1° try

↓ ACTH
2° try

∞ short sayan ACTH en to confirm Dx adrenal insufficiency

② Long Syan ACTH en in 3 days

→ ↑ ACTH endogenous / short sayan ACTH en not ↑
ACTH in 1° try or 2° try

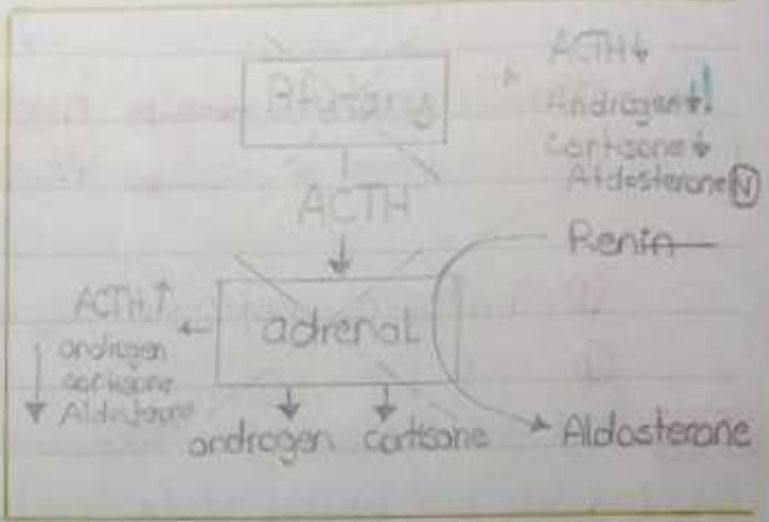
1° try

↓ cortisol

2° try

↑ cortisol

exogenous withdrawal



!! Shamatos & Autoimmune endocrine failure type II multiendocrine failure

Pituitary hypovastis

• Thyroides < Graves
Hashimoto's
• Pancreas B-cell (type I DM)

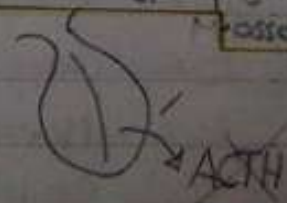
• Stomach affect Parietal cells
(Perineuritis areola)

• Intestine Ab affected not absorb
Glucose (celiac disease)

• Atrophy gonads (fertility & sub fertility)

• Melanocytes (depigmentation) in area
& hyper pigmentation

Passo & MG



بني
القول

(4)

Rx - ① cortisone $\downarrow$ $\rightsquigarrow$ hydrocortisone follow up by symptoms improvement by c/p
② Aldosterone $\downarrow$ $\rightsquigarrow$ fludrocortisone

Follow up by renin Level

③ Androgen not given any more

④ card Dx

Case :- Adrenal insufficiency on hydro & fludrocortisone syx & renin level (V)
Pt is (prepared for elective cholecystectomy) your action? $\uparrow$ dose of hydro & fludrocortisone stress condition

* **Acute adrenal failure** = as adrenal Hge / waterhouse fridrich \$

Saline NaCl normal saline

Sugar 10-20% Dextrose saline

Steroid 100 mg bolus IV

then 50-100mg during 6hr **slow infusion** even IM

key word of adrenal insuff. fatigue, epigastric pain, vomiting, hypotension, hypoglycemia

② anion gap

* **Pheochromocytoma** = rule of 10

enterochromaffin cell tumour found in ganglia (neuroendocrine tumour)

10% Bilateral

10% extra medullary

10% Malignancy

10% child onset

10% familial

90% sporadic

Adrenal medulla

Paraganglioma
(Paraspinal ganglia)

5

Neuroendocrine tumour

→ catecholamine (↑ sympathetic activity)

(Pulsatile tumour → symptoms in episodic attacks)

* C/P :-

CNS anxiety, unexplained

↑ sympathetic over activity

irritable

Headach

Sweaty = Diaphoresis = perspiration

Pupillary dilated

tremor

كلما يزيد sympathetic كلما يزداد وقت
ييزيد مدة حث Parasympathetic
في الاغصان الممتدة من القلب
Sympathetic

Palpitation • Abd pain

Pallor

Paradoxical effect

flushing

HTN

Paradoxical effect

Hypotension

Micturition

Parasympathetic

Paroxysmal Attacks of :-

Pressure HTN 90% of cases

Pain Headach 80%

perspiration sweaty 70%

palpitation 60%

Pallor

Pupillary dilation

→ symptoms & sign of
Panic attack.

* Whipple triad :- appear when insulin ↑ (case of hypoglycemia)

tremor

sweating

palpitation

if dextrose given & symptoms disappear
confirm → Whipple triad

To differentiate b/w Whipple & pheochromocytoma → give dextrose

* Ix of Pheochromocytoma

① 24hr urine collection measure catecholamine

may measure (Vanyl mandelic acid) → metabolism of adrenaline

directly catecholamine to confirm Dx → may be **blue high** (with 30% rich in K^+)

② CT scan Abdomen (Adrenal mass 90%)
extra adrenal 10%

③ MIBG methyl iodo benzyl guanidine highly absorbed by
enterochromaffine cells (best investigation of choice) show if case
develop metastasis or not

+ Rx- if localized in adrenal → surgery

according if metastasis or not operable

to MIBG
finding

give α -blocker → then
Vasoconstrictor (for 3 days)
after 3 day

now days give **Labetolol**

DOC of
(malignant HTN)

malignant HTN &
encephalopathy
nifedipine
(Ca^{2+} channel
blocker)

can cross B.B.B.
if given 1st α -blocker
free all catecholamine
work of α -blocker
bronchospasm → severe malignant
HTN sever cerebral Hge & death

* Association :-

MEN2

← multiple endocrine neoplasia → **MEN1**

- HOCM

- von Hippel Landau \$

- Sturge Weber \$

- Neurofibromatosis (cafe au lait spot)

MEN

asso Pheochromocytoma

كبيرة (HOCM) بيهلوا von Hippel لما يشوفوا

حاجات لون أحمر Sturge أو قهوي Neurofibromatosis

in midline I

• Pituitary

• PTH

• Pancreatic
(ZES)

II

A

• PTH
adenoma

B

• intestinal
neurema

• medullary carcinoma
of thyroid gland

• Pheochromocytoma

